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ABSTRACT

The head, neck and trunk kinematic responses of four volunteer test subjects, recorded during a series of experimental low velocity motor vehicle collisions, have been measured and analyzed. Using data obtained from multiple high speed film, video and electronic accelerometer measurements of the test subjects, it was found that the actual kinematic responses of the human head, neck and trunk that occur during low velocity rearend collisions are more complex than previously thought. Our findings indicate that the time-honored description of the cervical "whiplash" response is both incomplete and inaccurate.

Although the classic "whiplash" neck response to rearend collisions and the widely accepted hyperextension/hyperflexion cervical injury mechanism have been extensively written and speculated about, there have been little human experimental data available, especially for low velocity collisions. Low velocity collisions are defined in this report as motor vehicle collisions in which the impact related change of the rearended vehicle's velocity (ΔV) is about 12.9 kph (8 mph) or less. Throughout nearly 4 decades of experimental crash testing, low velocity mishaps (as defined above) have been felt to have a minor injury causation potential and have remained a relatively unstudied area. The absence of good experimental data, accurately defining real occupant kinematic response during this common type of traffic accident has spawned a plethora of divergent concepts, ideas and speculation about possible injury mechanisms.

In February 1991, a series of vehicle collision tests using fully instrumented volunteer human test subject/drivers and a Hybrid III manikin passenger was conducted, utilizing local testing facilities. This project was undertaken to better define human, dummy and vehicle responses during low velocity collisions.

METHODS

VEHICLES — Four test vehicles, a 1986 Dodge 600 convertible, a 1984 Buick Regal Limited coupe, a 1984 Ford Club Wagon van and a 1984 GMC 1500 pickup truck were prepared for the test protocol. Each vehicle was without evidence of collision structural damage and was in roadworthy condition with factory standard equipment and parts. The convertible and coupe had factory standard energy absorber bumper systems, and the truck and van bumper systems were rigidly mounted to the vehicle frame structure. Although each vehicle remained in stock condition to the maximum extent possible, the testing protocol required a number of modifications. The test subjects had to be photographically accessible, so the upper portion of the Ford van's left B-pillar and the front doors of each vehicle were removed. The rear window of the pickup truck was replaced with a small Lexan panel, and headrests in the convertible and the coupe were kept in the raised position. Several mounting points for high speed cameras were installed on each vehicle. Each vehicle's original, factory standard 3-point restraint system was used throughout the tests. Vehicles were checked prior to testing and any bumper assembly damage found was repaired with new parts.

TEST SUBJECTS — The proposed test protocol was evaluated by the University of Texas Health Science Center Institutional Review Board and IRB Protocol #9010099006 of the University of Texas Health Science Center, under DHHS Regulation 46.110(3), approved the use of four human test subjects selected from the staff of Biodynamic Research Corporation in the test series. Four healthy volunteer male test subjects, ranging in age from 45 to 56, completed pre-testing physical evaluations including radiographic imaging studies of the cervical, thoracic and lumbar regions of their spines. Test subject marking for photographic analysis included a stiff "U" shaped yellow rod attached to an individually fitted biteblock and accelerometer assembly and oriented rearward on both sides parallel to the aluminum strip connecting the accelerometers and the closed

mandible/maxilla. There was also a photographically visible mark placed below and slightly behind the left external auditory canal over the mastoid prominence as an approximation of the lateral projection of the upper end of the cervical spine. Reference marks were applied to the skin over the test subject's left neck, simulating the lateral projection of the cervical spine. Targets were placed approximately over the left gleno-humeral joint and lateral left elbow on a tight fitting garment worn over the torso and arms. A Hybrid III anthropomorphic test dummy was fitted with a biteblock type accelerometer assembly and had similar right side anatomical reference point markings applied, with the exception of the dummy's already exposed neck area.

INSTRUMENTATION — Each vehicle, had a triaxial LSCB-10 accelerometer array mounted on the vehicle frame to measure Gx (forward/rearward), Gy (right and left lateral), and Gz (upward/downward) motions and a biaxial accelerometer array (Gx and Gz) on the driver's side seatback. Contact switch operated flash units were installed in visible positions to allow photographic time marking of initial bumper to bumper contact and a similar contact switch cued the electronic data acquisition system. Additional instrumentation to accomplish other test objectives was also installed on the vehicles. Test subject instrumentation included a lightweight triaxial accelerometer assembly of Endevco #7290-30 and Endevco #7290-10 accelerometers mounted on a short aluminum strip fixed to an individually fabricated mouth piece (biteblock) which, when held with normal jaw closure pressure, allowed no appreciable relative motion between the accelerometer assembly and the test subject's maxilla/mandible. An identical accelerometer array mounted on an aluminum strip was affixed in an equivalent position on the Hybrid III manikin's head. A biaxial accelerometer assembly using similar sensors was affixed to a corset-like garment and was worn by one test subject during two of the test runs to measure Gx and Gz direction acceleration. Electronic data transfer during test runs was accomplished by a sliding loop umbilical bundle connected to a PAC-5800 high volume data acquisition system housed nearby.

PHOTOGRAPHIC EQUIPMENT — Photographic documentation of test runs was accomplished by several Redlake LoCam Model #51 high speed 16 mm cameras operated at 500 frames per second (nominal) and equipped with an LED timing light operating at 100 hertz. These cameras were mounted at various locations on the vehicles and from several fixed positions about the test site. One high speed video unit and several tripod mounted standard video cameras also recorded the events.

TEST SITE — The test site was established on a level section of a standard, asphalt paved roadway and had electronic speed trap instrumentation and high speed video tracking with near realtime velocity measurement capability. Impact speed reproducibility was achieved by the use of a specially constructed ramp permitting gravity acceleration of the striking vehicle. The striking vehicle's starting position on the ramp was calibrated before each test run to ensure that the resulting velocity at the impact point was in the desired range. The actual closure speeds and resulting changes in velocity of both the striking and the struck vehicles during the test runs were accurately determined by high speed film, high speed video and the electronic speed trap with satisfactory agreement. High speed cameras on the vehicles and at fixed site positions were actuated by a central electronic control and the video cameras were controlled by individual operators responding to auditory and visual cues.

TEST PROCEDURES — Test runs were conducted according to a protocol which tested a variety of combinations of vehicle to vehicle collisions and exposed test subjects to both striking and struck roles during tests planned for a forward struck vehicle ΔV of 4 kph (2.5 mph) and 8 kph (5.0 mph). The striking vehicle was backed up the ramp by the test subject-driver to a calculated position and released with the transmission in neutral and the engine running. It then rolled down the ramp and through the impact point speed trap where the velocity was recorded. This procedure was repeated until the desired impact speed was reproducibly achieved. The vehicle to be struck was then placed into its stationary position at the impact point. The striking vehicle then rolled down the ramp and over the level pavement to the impact point. No vehicle control inputs were made during test runs, except for minimal steering inputs to ensure centerline contact between vehicles and late braking after the test impact perturbations were over. In some cases, to prevent an over or underride situation, the height of one of the two vehicles was elevated by the use of wooden planking which formed an elevated roadbed. After each test collision, the driver's physical condition was checked, post-test photographic assessment of vehicle damage was completed, and electronic test result data storage was accomplished. Data from ten manned vehicle to vehicle test collisions were recorded.

HEAD, NECK AND TRUNK KINEMATIC RESPONSE ANALYSIS — The purpose of this article is to report our findings after a detailed analysis of human head, neck and trunk kinematic responses occurring during and immediately after low velocity rearend impacts. Relevant data included the recorded G-time information from the human and vehicle mounted accelerometers, displacement-time data taken from the high speed film record and the slow motion video record of each test subject's motion during the collision sequences. The electronic G measurement data was processed into a usable form with smoothing to eliminate noise artifacts and very short term transients. Biteblock G-time vector resultants were calculated from Gx, Gy and Gz data recorded for each collision sequence. In order to obtain true (earth orthogonal) G vector resultant data, mathematical coordinate transformation was done to account for the curvilinearly displaced path of the biteblock reference frame from its initial earth orthogonal orientation and required utilization of time-angle data obtained by measurement from the high speed film record. An additional series of mathematical manipulations gave an earth reference based G-time history for a point near the junction of the head and upper cervical spine. This information was correlated with point displacement information obtained from the high speed film by plotting each

test subject's reference points (biteblock, mastoid process, shoulder and hip) with respect to time, as well as the vehicle and background reference points using a precision optical digitizing process. Cervical extension and flexion angles were determined using digitized high speed film displacement data. The G-time and displacement-time data was then correlated and the results validated by a detailed comparison of the calculated data with the frame by frame video recordings of the impact related test subject kinematic responses.

RESULTS

TEST RELATED CLINICAL FINDINGS -- Each test subject had from 3 to 7 vehicle to vehicle test collision exposures, divided between the striking and struck roles during the 10 test collision series. (See Table 1.) These test runs were conducted during two weekend test periods separated by an eight day hiatus. No test subject reported having discomfort symptoms during or immediately after any of the test collisions. Test subject number 4 noted no symptoms at all related to his 6 test exposures. Beginning about 45 to 60 minutes after Test 2, test subject number 1 reported a "twinge" of discomfort at the posterior base of his neck which lasted about two hours. The discomfort was gone by the time of his participation in test number 3 and did not recur later. Test subject number 2 noted the onset of "achiness" in the paraspinal musculature at the base of his neck the morning of test day 12, after participating in a total of 6 test runs during the preceding two day test period. His symptoms lasted about 4-5 hours and resolved without recurrence. Test subject number 3 reported the onset of mild low and mid-neck discomfort over the area of his C6, C7 and T1 vertebra and discomfort in his trapezius musculature on the morning following his three test runs on day 10. The pain was gone the next day, but he continued to have mild discomfort on extreme neck extension and lateral flexion until it gradually resolved during the next three days. No treatment or therapy was needed and none of the test participants had any further symptoms that related to their test exposures for greater than eighteen months following the testing.

PHOTOGRAPHIC ANALYSIS OF AN EXAMPLE KINEMATIC RESPONSE — The following is a description of one test subject's kinematic responses to a typical test (Run 7), as he experienced a forward ΔV of 7.83 kph (4.87 mph). (See Figure 1.)

Phase 1 - Initial Response (0 to 100 milliseconds) — During the first 50 milliseconds after bumper impact, the subject's entire body appeared motionless with reference to the test site background, while the vehicle and driver's seat moved forward about 5 to 8 centimeters (2 to 3 inches). At about 60 milliseconds after contact, the lower part of the seatback cushion had become compressed enough to begin moving the test subject's hips and low back forward and upward (See Figure 2). At the same time, the upper part of the seatback was flexing rearward with respect to the vehicle and the seatback cushion was being compressed on the test subject's still nearly stationary upper torso.

Phase 2 - Principal Forward Acceleration (100 to 200 milliseconds) — At about 100 milliseconds, the seatback had nearly reached its maximum rearward flexed position of about 10 degrees past its normal position, and the test subject's upper trunk had begun movement forward and upward. The

Run No.	Test Subj No.	Struck Vehicle Type	ΔV (kph)	Test Subj No.	Striker Vehicle Type	ΔV (kph)	Day No.
1	2	Van	3.48	1	Convert	-4.81	1
2	1	Van	6.45	4	Pickup	-6.04	2
3	1	Pickup	3.04	4	Van	-3.35	2
4	4	Pickup	6.65	1	Van	-6.74	2
5	3	Convert	na	2	Coupe	na	10
6	3	Convert	8.06	2	Coupe	-7.82	10
7	2	Coupe	7.83	3	Convert	-9.24	10
8	2	Van	6.61	4	Pickup	-8.21	10
9	2	Coupe	3.93	4	Pickup	-3.28	11
10	4	Pickup	7.03	2	Van	-7.48	11

Table 1. Test Subject Driven Low Velocity Collision Test Series

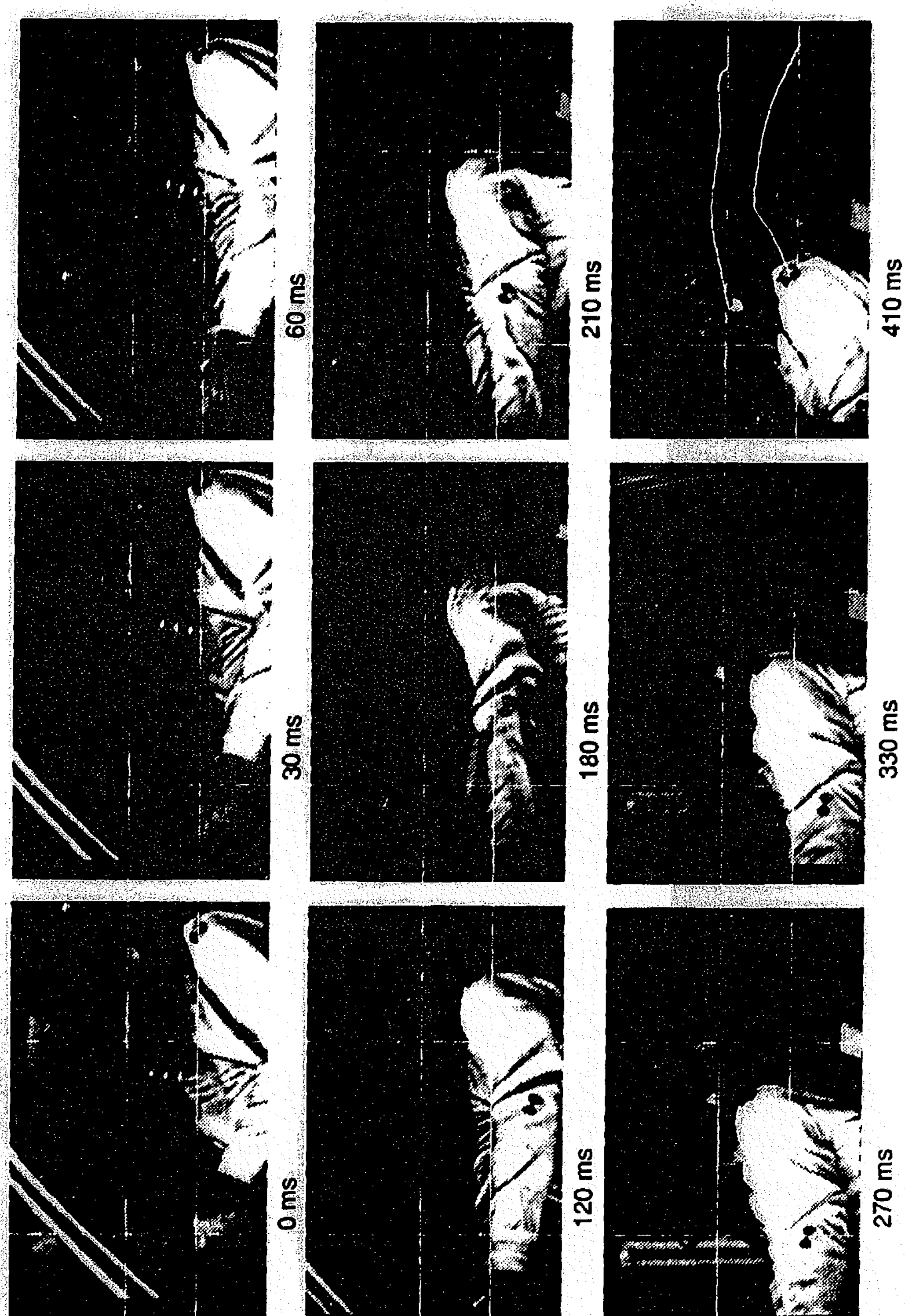

Figure 1. Example Response to Rear Impact - Test 7
Note. Grid lines are earth fixed. Last picture shows time trace of neck top and shoulder pathways.

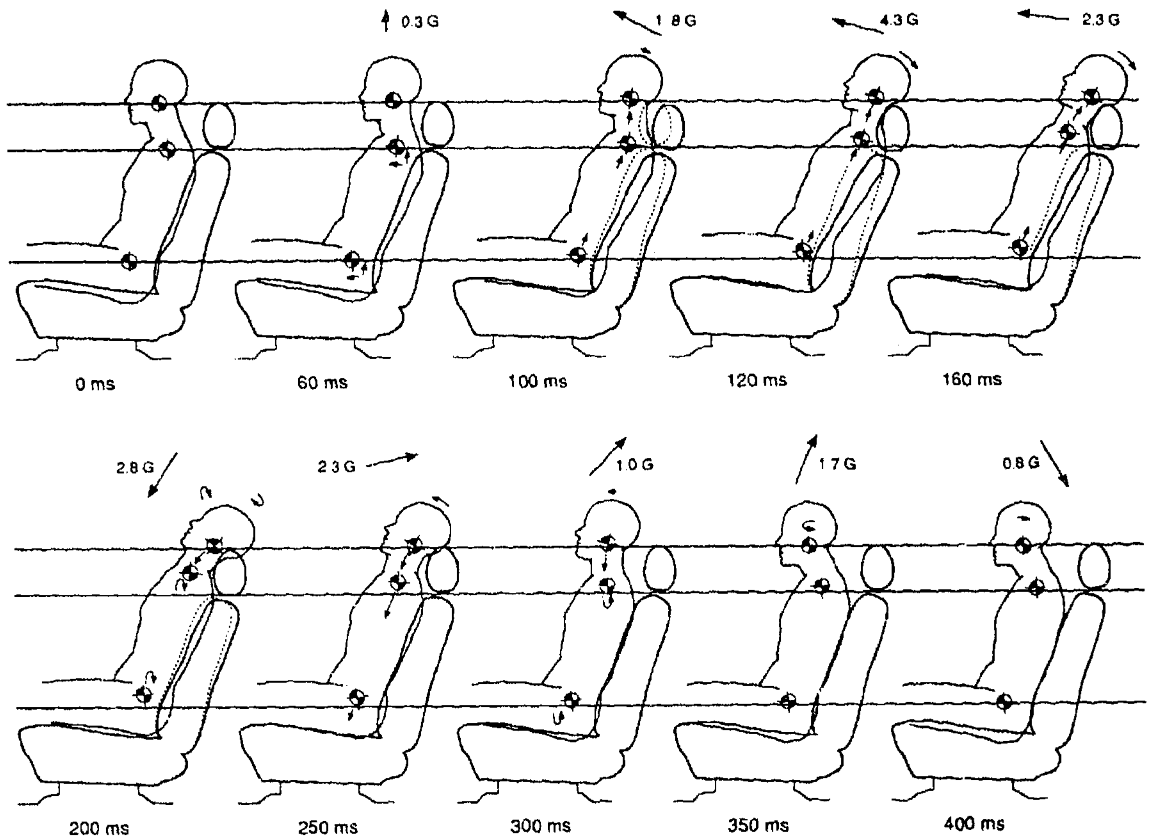

Figure 2. Head, Neck and Trunk Responses to Low Velocity Rear End Impact

subject's head and neck were still almost stationary with respect to the earth until about 120 milliseconds when, as the subject's hips and trunk rose upward on a path parallel to the rearward flexed seatback, his neck appeared to be axially compressed and straightened as the top of the cervical spine began moving upward and rearward with respect to the forward moving vehicle. Subsequently, the subject's head began a biteblock upward and rearward rotating movement with respect to the subject's shoulders. By 160 milliseconds the forward and upward movement of the subject's ascending upper torso had begun to pull the base of his neck forward into apparent tension and starting the forward motion of the subject's head, even as his occiput continued to tip downward towards the seat headrest.

<u>Phase 3 - Head Overspeed/Torso Recovery (200 to 300 milliseconds)</u> — At 200 milliseconds the upward motion of the subject's trunk and shoulders had ceased after about 9 centimeters (3.5 inches) of rise and the extension and rotational angulation of his head had stopped about 45 degrees rearward from vertical. The subject's top of the cervical spine marker point had risen about 1.25 centimeters (0.5 inches) above its initial vertical position and the subject's head was starting to reverse its motion, with respect to the vehicle, into a forward arcing movement. By 250 milliseconds the forward rebounding head had not yet reached vertical, but the subject's trunk, neck and head were already descending along a path parallel to the seatback. His trunk was nearly halfway towards its starting position with respect to the seat bottom. By this time, the seatback had returned to its pre-impact normal angle and the subject's torso was rebounding forward and away from the seatback's surface. At this point the test subject's upper body was probably being actively retarded by the tightened restraint system. The restraint system had become more than normally tightened when the spring powered seatbelt retractor reeled in about 5 to 8 centimeters (2 to 3 inches) of seatbelt and shoulder harness slack that had

been produced by the initial compression and relatively rearward flexion of the forward moving seatback cushion by the stationary test subject's torso. This autotightening phenomenon was noted consistently in several vehicles during our test series.

Phase 4 - Head Deceleration/Torso Rest (300 to 400 milliseconds) — After 300 milliseconds from first bumper contact, the descent of the test subject's trunk had been completed and his trunk was moving at essentially the same velocity as the vehicle. The rebound forward motion of his head, now positioned near vertical, continued but was being actively decelerated by the tension in his neck. At about 400 milliseconds his head had reached its most forward position with respect to the vehicle. The biteblock to mastoid reference line was nearly level with the horizon and the head was slowing to a nearly level, forward and lowered position with respect to the subject's shoulders. The subject's head then began a return movement relatively rearward and upward towards a normal upright position over his shoulders.

Phase 5 - Restitution Phase (400 to 600 milliseconds) — After about 450 milliseconds after first bumper contact, all test subject body parts were traveling at approximately the vehicle's velocity and his immediate impact related motions were nearly completed. During this period the test subject's head and biteblock returned to approximately their pre-test positions with respect to the vehicle. However, the test subject's shoulders and hips rested in a position about 3.8 centimeters (1.5 inches) higher than before.

TIME-DISPLACEMENT AND ACCELERATION ANALYSIS — Analysis of the time-displacement record of the body reference targets and the G-time record of the biteblock sensors, referenced by calculation to a point approximately at the top of the cervical spine, defined the x direction acceleration profile for each test. Analysis also confirmed the existence of significant upward and downward accelerated motion of the trunk, neck and head along the z axis. Vehicle and test subject motions along the y axis were not found to be significant in this test series.

Test subject cervical extension and flexion angles observed during this test series were always found to fall within the subject's voluntary physiological limits. Hyperextension or hyperflexion did not occur during any of the test runs. The maximum cervical extension (nose up) observed for all struck vehicle test runs appeared to be self limited to a maximum of about 40 to 45 degrees, even for test runs using the van, which had no headrests. Rebound cervical flexion was minimal for every test with head angulation (nose down) from the normal upright posture averaging three to five degrees or less, as the typical test subject's head followed a mild, head level, controlled forward and downward decelerative motion, followed by a gentle return to a normal upright posture. Throughout their impact related kinematic responses, each test subject's head and torso generally remained within 13 to 18 centimeters (5 to 7 inches), or less, of their pre-impact positions with respect to the vehicle.

Figure 3 shows Test Run 7 resultant accelerations acting at the top of the cervical spine for selected times, beginning 60 milliseconds after bumper contact (time 0). Figure 2 also shows these G vector resultants.

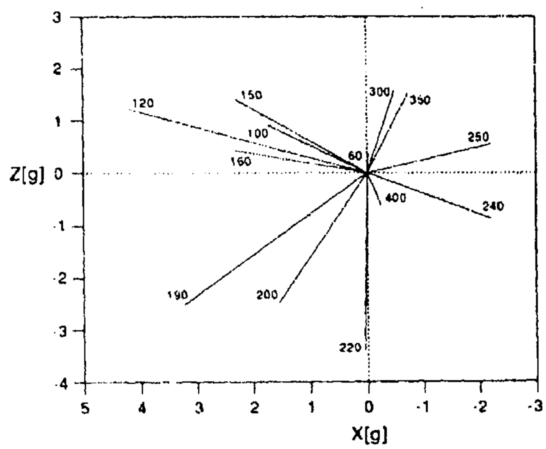

Figure 3. Resultant Acceleration (Gs) of the Neck Top at Selected Times after Impact (milliseconds). Test 7

Phase 1 - Initial Response (0 to 100 milliseconds) — The initial top of the cervical spine resultant acceleration at about 60 milliseconds after contact is upward at about 0.3 G and soon rises to 1.8 G upward and forward by 100 milliseconds.

Phase 2 - Principal Forward Acceleration (100 to 200 milliseconds) — At about 120 milliseconds acceleration forward and upward climbs to an overall peak of about 4.3 Gs. As forward Gx acceleration continues, the top of the cervical spine Gz acceleration reverses from a +1.2 Gz positive (upward) peak at 150 milliseconds to a peak negative (downward) -3.5 Gs at about 220 milliseconds. At 190 milliseconds resultant acceleration at the top of the cervical spine is nearly 4 Gs in a downward and forward direction.

Phase 3 - Head Overspeed/Torso Recovery (200 to 300 milliseconds) — At 200 milliseconds, the subject's head begins to arc forward with respect to his torso and over the next 30 milliseconds the top of the cervical spine is being accelerated forward and downward at 2 to 3.5 G. Simultaneously, the torso begins to return forward and down the seatback slope. By 220 milliseconds most of the forward acceleration of the top of the cervical spine has been accomplished and the actual upward motion of the torso and head has nearly ceased. Also by this time, the acceleration altered seatback angle has nearly returned to its normal position and the seat springs have become almost unloaded. By 250 milliseconds, the forward moving torso is being retarded by the restraint system and the Gz acceleration of the

top of the cervical spine is reversing as the trunk is now about halfway down the seatback and moving towards its original position. The subject's head has now arced forward with respect to the torso, decreasing its rearward angle to about half the maximum angle achieved and the top of the cervical spine is being accelerated rearward and slightly upward at about 2.3 G. Over the next 50 milliseconds, as the seat cushion is compressed by the subject's downward moving body, the upward Gz acceleration experienced at the top of the cervical spine increases to about 1.5 G.

Phase 4 - Head Deceleration/Torso Rest (300 to 400 milliseconds) — Around 300 milliseconds the downward motion of the trunk reverses and the subject's head has returned to about its pre-impact position over his torso. At this point, the resultant acceleration at the top of the cervical spine decreases to about 1 G directed upward and rearward. During the next 50 to 100 milliseconds, the trunk and torso begin to reassume a rest position. At the same time, the more rapidly forward moving head continues forward and downward while maintaining a nearly level position with respect to the horizon. During this maneuver the lower neck becomes mildly flexed while the top of the cervical spine mildly extends resulting in a kind of "level head bob". At around 400 milliseconds the subject's head begins a controlled return upward and rearward while still remaining nearly level. The resultant upward and rearward acceleration at the top of the cervical spine at this point is 1.7 G.

Phase 5 - Restitution Phase (400 to 600 milliseconds) — After 400 milliseconds the test subject's body position returns to about its pre-impact position with the exception of hip and shoulder positions about 3.8 centimeters (1.5 inches) higher than at the start of the test. This may have resulted from shifting of trunk and hips on the seat's surface, residual spinal curvature straightening, or perhaps due to lack of time for the seat cushions to fully conform to their recently reacquired load. Between about 400 and 600 milliseconds after initial contact, mild Gx and Gz oscillations appear to briefly continue but are rapidly damped and soon blend into a low level "jiggle", probably associated with the vehicle's movement over the pavement.

GENERALIZED FINDINGS

The kinematic responses of the struck vehicle test subjects for each test run were qualitatively similar to the above description. There were some subtle but consistently observed differences in the kinematic responses of test subjects due to the dissimilar design between the coupe/convertible seats and the more upright and somewhat stiffer seats of the pickup truck and van. The test subject's kinematic responses for the 4 kph (2.5 mph) ΔV test runs were similar to the 8 kph (5.0 mph) ΔV categories but visibly reflected the four-fold decrease in collision related energy. Lower velocity test runs, in general, were associated with considerably lessened overall kinematic activity and much milder cervical extensions. From a clinical standpoint, the nominal 8 kph (5 mph) ΔV test runs appeared to be on the threshold for mild cervical strain injury for our repetitively exposed test subjects. The nominal 4 kph (2.5 mph) ΔV test runs were considered later by the participating physician test subjects to have been so very mild that a single exposure would have been unlikely to have resulted in any symptomatology.

DISCUSSION

In reviewing the voluminous literature on this subject, especially the many articles on clinical analysis, experimental testing and computer based modeling efforts, one must be careful about making the assumption that the conclusions about human head and neck kinematics reached in these studies necessarily apply to low and very low velocity rearend collisions involving "real people". The majority of these studies have been primarily based on higher speed, 24 to 80 kph (15 to 50 mph) or more, rearend collisions utilizing dummies, cadavers, animals, computer models and very few live volunteers.

The puzzling, and sometimes stubbornly persistent clinical symptom picture frequently associated with "whiplash" injury, especially in the absence of objective physical findings, leads by default to the assumption that something unknown is happening during low velocity rearend collisions which is damaging cervical structure(s) without causing objectively discernable acute changes. Since exaggerated neck motion far beyond tolerable human limits had been frequently observed in dummies and cadavers during high speed testing, it has been commonly assumed that cervical hyperextension and hyperflexion would also occur during low and very low velocity collisions. It has been conjectured by many that the forced movement of the neck beyond physiologic limits was the injury mechanism causing the "whiplash" syndrome, especially in thin necked, unprepared people.

The test subjects in this series were robustly healthy middle-aged men who were well aware of testing procedures. To the maximum extent possible, test subjects were kept unaware of the specific time of the impending test impact and all consciously attempted to maintain a normal relaxed muscle tone while awaiting the impact. While no test subject exceeded his normal voluntary range of cervical motion during the test runs, three of the four did subsequently develop transient, mild cervical strain symptoms which indicated that there was an injury mechanism that was not dependent upon exceeding the physiologic limits of cervical motion.

There has been an occasional reference in the reviewed literature to vertical movement of the rearended vehicle test subject (usually a dummy) under specific test circumstances, or as a possibility in conceptual modeling. However, since forward acceleration becomes so dominant in the often studied higher speed rearend collision, analysis of test subject vertical motion has been generally ignored. We were conceptually aware before the test series of the possibility of impact-related acceleration in the Gz direction and made provisions to measure it. However, the magnitude and apparent causes of the vertical movement associated with our human test subjects and the corresponding lack of a

similar vertical excursion observed in the Hybrid III dummy had not been predicted from our review of the subject literature.

Observation of the high speed film record of the struck vehicle test runs disclosed that the test subject's early upward neck movement and initial forward head motion were due to several probable mechanisms. First, there was an upward lofting action caused by the normal vehicle seatback angle, which was itself increased by acceleration related deflection of the seatback. Second, there appeared to be an acceleration related straightening of the normal thoracic, cervical and, perhaps, lumbar spinal curvatures against the forward moving seatback surface, resulting in an apparent axial lengthening of the spine. Third, resistance to neck structure bending generated by the normal postural muscle tone of the upright neck may contribute to the initial forward head acceleration as recorded at the top of the cervical spine.

The upward movement of the test subject's trunk and neck base caused a more complex than expected motion of the neck and head. This initial upward motion was immediately followed by a sudden and surprisingly vigorous descent of the trunk, which may have resulted from a "rubberband" effect of the kinematically straightened and stretched trunk/spinal structures as the upwardly moving pelvis becomes restricted by the lapbelt. Shortly after the subject's trunk reaches the limit of upward compliance permitted by the lapbelt, the spine appeared to forcefully return to its original unstretched and curved state.

According to our measurements of the struck vehicle test subjects during the 6 to 8 kph (4 to 5 mph) ΔV test runs, there was an initial axial compression, measured at the top of the cervical spine, which accelerated the head at about 1 to 1.5 Gs upward, followed, as the base of the neck was pulled forward and downward, by a brief peak axial tension through the rearward extended cervical spine of about 2 to 4.5 Gs. The less energetic 3 to 4 kph (1.9 to 2.4 mph) ΔV test runs resulted in essentially the same type of head and neck motion with much decreased accelerations and kinematic responses.

At the low and very low velocity rearend collisions evaluated during our test series, the most likely injury to have occurred was a mild cervical and upper shoulder muscular strain. In our experience, this relatively mild injury would have been expected to be, and was, self-limited. It appears from our present results that the principal mechanical stress on the cervical spine related to rearend vehicle collisions in the low to very low velocity range is a rapid compression-tension cycle directed axially through the cervical spine and neck musculature as the neck sequentially compresses, extends, accelerates the head in tension and then flexes, all occurring within the normal physiologic range of motion limits of the neck. A suggested probable injury causation mechanism consistent with these observations would be a localized cervical/upper thoracic muscular strain caused by rapid, short excursion, forced muscular compliance to sudden unanticipated tension, occurring at an onset rate beyond the physiologic tolerance of the affected muscles' intracellular micro-structures. Belated, overactive or out of synchronization reflex muscular tensioning to control head motion may contribute to the muscular strain process, although our test subjects muscular corrective mechanisms appeared to be appropriately coordinated and effective by about 200 to 250 milliseconds after impact. An additional, less likely, but possible injury mechanism may be simple compression-related acute micro-contusional injuries to the cervical and upper thoracic spine connective tissue and joint structures. Similar compression-tension injury causation mechanisms may account for the mild low back discomfort symptoms that are sometimes reported by individuals involved in low velocity rearend collisions.

Photographic observation of the head, neck and upper torso relationships that occurred during each test subject's peak cervical extension suggested the possibility of an inherent, anatomically based, neck extension limitation mechanism. A triangular shaped support structure for the top of the cervical spine is formed by the supporting structures of the upper torso, the partially extended (straightened) cervical spine and the muscles of the anterior neck that normally maintain the head erect. Initial cervical muscle bracing would involve pre-existing normal neck muscle tone, followed during the middle and end of the initial kinematic response period by reflex and voluntary muscular head righting activity. This progressively more active bracing mechanism, in conjunction with the downward and forward movement of the shoulders and base of the neck, (which occurred in our test series well before the subject's head and neck had reached full extension), may help self-limit (at least in the low velocity situation) neck extension to within physiologically tolerable limits. The absence of this active bracing mechanism in both anthropomorphic dummies and cadaver test subjects may account for the observation of unchecked rearward head motion and neck hyperextension reported in experiments utilizing these imperfect human analogs as test subjects.

CONCLUSIONS

The data from our low velocity rearend collision test series using volunteer test subjects supports the preliminary conclusion that substantial Gz direction acceleration occurs and is associated with both compressive and tensile forces sequentially directed axially through the cervical spine.

These push-pull forces probably represent an injury causation mechanism independent of the commonly described cervical "whiplash" hyperextension/hyperflexion mechanism. For rearend collisions within the velocity range included in our test series, the classic "whiplash" injury mechanism, seems unlikely since no hyperextension or hyperflexion was observed in any of our test subjects. Despite having experienced no neck excursions beyond their voluntary range limits, three of our four test subjects transiently had very mild, but clinically classic neck discomfort symptoms.

During the lower energy level 4 kph (2.5 mph) ΔV test runs, the test subject's relatively rearward head motion was similar but much milder and, in each case, the back of the test subject's head did not reach the headrest. The injury

causation potential during these tests was subjectively judged by the physician test subjects to have been minimal or non-existent.

The reported results of this low velocity test series suggest a compression-tension injury causation mechanism which probably can cause self-limited minor cervical, thoracic and lumbar muscle strains and, possibly, connective tissue and/or vertebral joint micro-contusional injuries and that may account for the discomfort symptoms commonly reported after low velocity rearend collisions. The very mild discomfort symptoms experienced by our three test subjects, after multiple test exposures, indicated that the 6 to 8 kph (4 to 5 mph) struck vehicle ΔV test conditions were probably at, or near, typical human threshold for very mild, single event musculoskeletal cervical strain injury.

The test results from our small number of test runs and relatively homogeneous test panel should be supplemented by further testing. This testing, which should include a wider variety of test subjects arranged in different seating positions, various riding postures and restraint system usage, would better define the complete range of expected kinematic responses by the vehicle riding general public to low and very low velocity rearend collisions.

BIBLIOGRAPHY

Ameis, Arthur. Cervical Whiplash: Considerations in the Rehabilitation of Cervical Myofascial Injury. Can. Fam. Physician, Vol. 32, 1871-1876 (September 1986).

Balla, John I. The Late Whiplash Syndrome. Aust. N.Z. J. Surg., Vol. 50, No. 6, 610-614 (December 1980).

Berton, R.J. Whiplash: Tests of the Influential Variables. Society of Automotive Engineers, 680080, 1-10 (January 1968).

Billig, Harvey E., Jr., M.D., F.I.S.C. The Mechanism of Whiplash Injuries. International Record of Medicine, 169, 3-7 (1956).

Bogduk, Nikolai, Ph.D., Dip. Anat. The anatomy and pathophysiology of whiplash. Clinical Biomech, 1, No. 2, 92-101 (1986).

Carlsson, Gerd, Nilsson, Stefan, Nilsson-Ehle, Anna, Norin, Hans, Ysander, Lars and Ortengren, Roland. Neck Injuries in Rear End Car Collisions. BioMechanical Considerations to Improve Head Restraints. American Association for Automotive Medicine, The Quarterly/Journal, 25-30 (July 1985).

Clemens, H.J., Burow, K. Experimental investigation on injury mechanisms of cervical spine at frontal and rear-front vehicle impacts. Proceedings of the Sixteenth Stapp Car Crash Conference, (1972); 76-104.

Crow, H.E. Injuries to the Cervical Spine, Paper presented at the Meeting of the Western Orthopaedic Association, San Francisco (1928).

Frankel, V.H. Temperomandibular joint pain syndrome following deceleration injury to the cervical spine. Bull Hosp. Joint Dis. (1965) 26:47-51.

Gay, James R., M.D., Abbott, Kenneth H., M.D. Common Whiplash Injuries of the Neck. J.A.M.A., Vol. 152, No. 18, 1698-1704 (August 29, 1953).

Gotten, Nicholas, M.D. Survey of One Hundred Cases of Whiplash Injury After Settlement of Litigation. J.A.M.A., 162(9), 865-7 (October 1956).

Harris, W.H., Hamblen, D.L., and Ojemann, R.G. Traumatic disruption of cervical intervertebral disc from hyperextension injury. Clin. Orthop., 60: 163-167, (1968).

Hirsch, Stuart A., M.D., Hirsch, Paul J., M.D., Hiramoto, Harlan, M.D., and Weiss, Andrew, M.D. Whiplash Syndrome Fact or Fiction? Orthopedic Clinics of North America, Vol. 19, No. 4, 791-5 (October 1988).

Hohl, M. Soft-tissue injuries of the neck in automobile accidents. J. Bone Joint Surg., 56A; 1675-1682, (1974).

Howard, Richard P., M.D., M.S., Benedict, James V., Ph.D., M.D., Raddin, James H., Jr., M.D., S.M., and Smith, Harry L., Ph.d, M.D. Assessing Neck Extension/Flexion as a Basis for Temporomandibular Joint Dysfunction. J Oral Maxillofac Surg, Vol. 49, 1210-1213 (1991)

Kallieris, D., Mattern, R., Miltner, E., Schmidt, Gg. and Stein, K. Considerations for a Neck Injury Criterion. University of Heidelberg, 401-417 (1991).

La Rocca, Henry, M.D. Acceleration Injuries of the Neck. Clinical Neurosurgery, 25, 209-17 (1978).

McKenzie, J.A. and Williams, J.F. The Dynamic Behavior of the Head and Cervical Spine During 'Whiplash'. J. Biomechanics 4, 477-490 (1971).

Macnab, I. Acceleration-extension injuries of the cervical spine. American Academy of Orthopaedic Surgeons. Symposium on the Spine, St. Louis: Mosby (1969), 10-17.

Macnab, I. The whiplash syndrome. Clin. Neurosurg (1973); 20:232-41.

Macnab, Ian, M.B., Ch.B., F.R.C.S. The "Whiplash Syndrome". Orthopedic Clinics of North America, Vol. 2, No. 2, 389-403 (July 1971).

Macnab, Ian, M.B., Ch.B., F.R.C.S. Acceleration Injuries of the Cervical Spine. The Journal of Bone and Joint Surgeries, Vol. 46-A, No. 8, 1797-1799 (December 1964).

Martinez, J.L, Garcia, D.J. A model for whiplash. J. Biomech (1968); 1:23-32.

Mertz, H.J. and Patrick, L.M. "Investigation of the Kinematics and Kinetics of Whiplash" Proceedings of the 11th Stapp Car Crash Conference, pp 175-206, SAE Paper 670919, (1967).

Mertz, H.J. and Patrick, L.M. Strength and Response of the Human Neck. Proceedings of the 15th Stapp Conference, pp 207-255, SAE Paper 710855, (1971).

Mertz, H.J. "Neck Injury in Biomechanics and its Application to Automotive Design" SAE, Warrendale, Publication No. P. 49, (January 1973).

Muzzy III, WH, Seemann, MR, Willems, GC, Lustick, LS, Bittner, AC: The Effect of Mass Distribution Parameters on Head/Neck Dynamic Response, Proc. 30th Stapp Car Crash Conf. Society of Automotive Engineers, Warrendale, PA, pp 167-184, (1986).

Norris, S.H. and Watt, I. "The Prognosis of Neck Injuries Resulting from Rear-end Vehicle Collisions" Journal of Bone and Joint Surgery, Vol. 65-B, No. 5, 608-11 (November 1983)

O'Neill, B., et. al. "Automobile Head Restraints - Frequency of Neck Injury Claims in Relation to the Presence of Head Restraints" A.J.P.H., 399-406 (March 1972).

Pearce, J.M.S. Whiplash injury: a reappraisal. Journal of Neurology, Neurosurgery, and Psychiatry, 52, 1329-1331 (1989).

Pietrobono, R. The anatomy of the cervical region with particular reference to the symptomatology of the 'whiplash' lesion. J. Bone Joint Surg. (1956); 38A; 438.

Pintar, FA, Sances, A, Yoganandan, N, Reinartz, J, Maiman, DJ, Suh JK, Unger, G, Cusick JF, Larson, SJ: Biodynamics of the Total Human Cadaveric Cervical Spine. Proc. 34th Stapp Car Crash Conf. Society of Automotive Engineers, Warrendale, PA, pp 55-72, (1990).

Prasad, P., et. al. "Dynamic Response of the Spine During -GX Acceleration" Proceedings of the 19th Stapp Conference, pp 869-897, SAE Paper No. 751172.

Raney, A.A., Raney, R.B., and Hunter, C.R. Chronic posttraumatic headache and the syndrome of cervical disc lesion following head trauma. J. Neurosurg., 6: 458-465, (1949).

Sances, Jr., et. al. "Experimental Studies of Brain and Neck Injury" Proceedings of the 25th Stapp Conference, pp. 149-194, SAE Paper No. 811032 (1981).

Sances, Jr., et. al. "Pathophysiology of Cervical Injuries" Proceedings of the 26th Stapp Conference, pp. 41-70, SAE Paper No. 821153, (1982).

Severy, D.M., Mathewson, J.H., and Bechtol, C.O., M.D. Controlled Automobile Rear-End Collisions, an Investigation of Related Engineering and Medical Phenomena. Canadian Services Medical Journal, VII, 727-59 (1955).

Snyder, R.G. "Human Impact Tolerance" 1970 International Automobile Safety Conference Compendium, pp. 712-782, SAE, Warrendale, Paper No. 700398, (1970).

States, J.D., Korn, M.W., Masengill, J.B. "The Enigma of Whiplash Injuries" Proceedings of the 13th Conference of the American Association for Automotive Medicine, pp. 83-108, AAAM, Arlington Heights, Illinois, (October 1969).

States, J.D., et. al. "Injury Frequency and Head Restraint Effectiveness in Rear-End Impact Accidents" Proceedings of the 16th Stapp Car Crash Conference, pp. 228-257, SAE, Warrendale, Paper No. 720967, (November 1972).

Thibault,L., Gennarelli, T.A. Biomechanics of Diffuse Brain Injuries. Proceedings of the 10th International Technical Conference on Experimental Safety Vehicles, Oxford, England, July 1-4, 1985. Washington, D.C., U.S. Department of Transportation, National Highway Traffic Safety Administration. DOT-HS-807-481, March 1989.

Thiemeyer, John S., Jr., M.D., Duncan, George A., M.D., Hollins, George G., Jr., M.D. Whip-Lash Injuries of the Cervical Spine. Virginia Medical Monthly, Vol. 85, 171-4 (April 1958).

Webb, Michael N., Terrett, Allan G.J. Whiplash: Mechanisms and Patterns of Tissue Injury. Journal of the Australian Chiropractor's Association, Vol. 15, No. 2, 60-69 (June 1985).

Weinberg, Simon, DDS, FRCD(C) and LaPointe, Henry, DDS. Cervical Extension/Flexion Injury (Whiplash) and Internal Derangement of the Temporomandibular Joint. J Oral Maxillofac. Surg, 45, 653-656 (August 1987).

White, A.A., III & Panjabi, M.M. "Clinical Biomechanics of the Spine". Lippincott, Philadelphia (1978).

Williams, J.F., McKenzie, J.A. Technical Notes. The Effect of Collision Severity on the Motion of the Head and Neck During 'Whiplash'. J. Biomechanics, Vol. 8, 257-259 (1975).

Wismans, J., Oorschot, H., Woltring, HJ: Omni-Directional Human Head-Neck Response. Proc. 30th Stapp Car Crash Conf. Society of Automotive Engineers, Warrendale, PA, pp 313-325, (1986).